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RESET-SLE™: Clinical Trial Evaluating Rese-cel (Resecabtagene Autoleucel), a Fully Human, Autologous 4-1BB Anti-CD19 CAR T Cell Therapy in Non-Renal SLE and Lupus Nephritis

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RESET SLE™

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Various risks, uncertainties and assumptions could cause actual results to differ materially from those anticipated or implied in our forward-looking statements. Such risks and uncertainties include, but are not limited to, risks related to the success, cost, and timing of our development activities and clinical trials, risks related to our ability to demonstrate sufficient evidence of safety, efficacy and tolerability in our clinical trials, the risk that the results observed with the similarly-designed construct, including, but not limited to, dosing regimen, are not indicative of the results we seek to achieve with rese-cel, the risk that signs of biologic activity or persistence may not inform long-term results, risks related to clinical trial site activation or enrollment rates that are lower than expected, risks that modifications to trial design or approach may not have the intended benefits and that the trial design may need to be further modified; our ability to protect and maintain our intellectual property position, risks related to our relationships with third parties, uncertainties related to regulatory agencies' evaluation of regulatory filings and other information related to our product candidates, our ability to retain and recognize the intended incentives conferred by any regulatory designations, risks related to regulatory filings and potential clearance, the risk that any one or more of our product candidates will not be successfully developed and commercialized, the risk that the results of preclinical studies or clinical studies will not be predictive of future results in connection with future studies, risks related to volatile market and economic conditions and our ability to fund operations and continue as a going concern. New risks and uncertainties may emerge from time to time, and it is not possible to predict all risks and uncertainties. Except as required by applicable law, we do not plan to publicly update or revise any forward-looking statements contained herein, whether as a result of any new information, future events, changed circumstances or otherwise. Although we believe the expectations reflected in such forward-looking statements are reasonable, we can give no assurance that such expectations will prove to be correct. Accordingly, you are cautioned not to place undue reliance on these forward-looking statements. No representations or warranties (expressed or implied) are made about the accuracy of any such forward-looking statements. For a discussion of these and other risks and uncertainties, and other important factors, any of which could cause our actual results to differ materially from those contained in the forward-looking statements, see the section entitled "Risk Factors" in our most recent annual report on Form 10-K and quarterly report on From 10-Q, as well as discussions of potential risks, uncertainties, and other important factors in our other filings with the Securities and Exchange Commission. Certain information contained in this Presentation relates to or is based on studies, publications, surveys and other data obtained from third-party sources and the Company's own internal estimates and research. While the Company believes these third-party sources to be reliable as of the date of this Presentation, it has not independently verified, and makes no representation as to the adequacy, fairness, accuracy or completeness of, any information obtained from third-party sources. The Company is the owner of various trademarks, trade names and service marks. Certain other trademarks, trade names and service marks appearing in this Presentation are the property of third parties. Solely for convenience, the trademarks and trade names in this Presentation are referred to without the ® and TM symbols, but such references should not be construed as any indicator that their respective owners will not assert, to the fullest extent under applicable law, their rights thereto.

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Disclosures

Author		Disclosures
Saira Sheikh		GlaxoSmithKline, AstraZeneca, Biogen, Cabaletta Bio, Aurinia Pharmaceuticals
Vimal Derebail		Novartis, Travere, Bayer, Forma Therapeutics, Merck & Co, Amgen, iCell Gene Therapeutics, Vera Therapeutics
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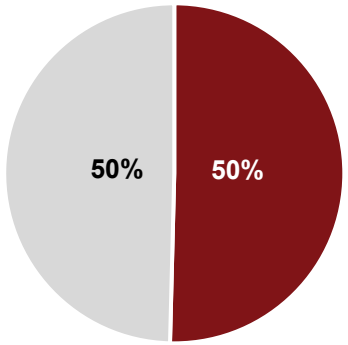
Durable Remission is Rarely Achieved in Lupus with Standard of Care[1]

Lupus is associated with poor outcomes, requiring long-term and burdensome immunomodulatory therapy[2–6]

Systemic lupus erythematosus

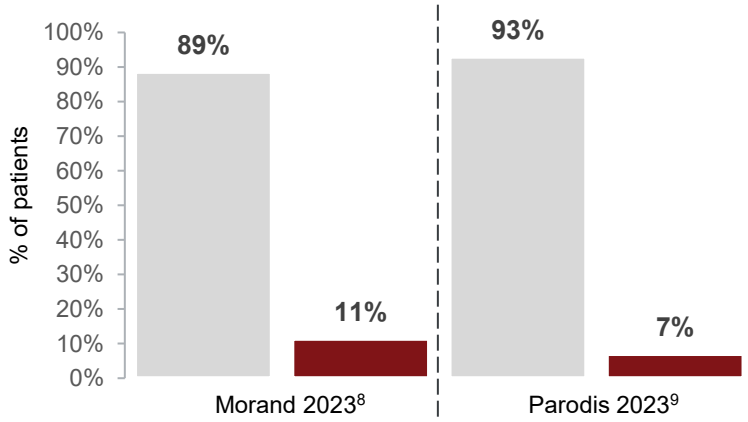
SRI-4 achieved by ~50% of patients across all treatment arms in biologic RCTs[7]



Petri 2022[7]

- Non-responders (1 year)
- Responders (1 year)

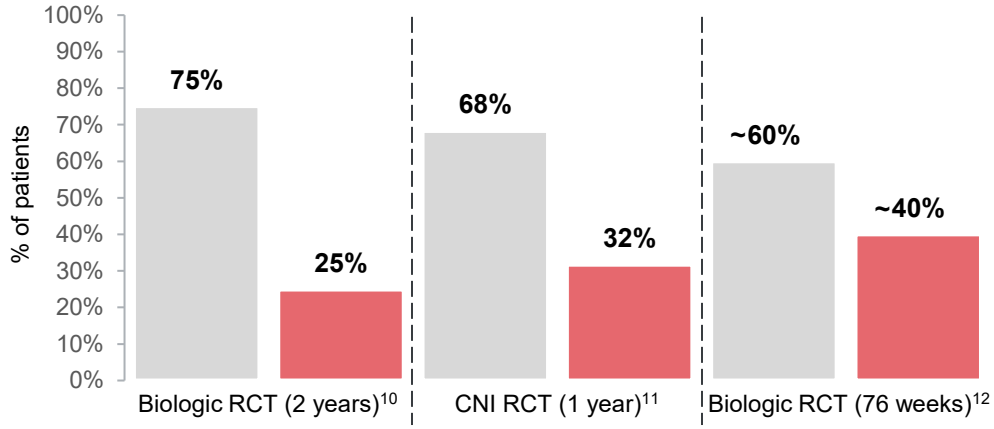
DORIS achieved by <12% of patients across all treatment arms in biologic RCTs



- Remission not achieved (1 year)
- Remission achieved (1 year)

Lupus nephritis

CRR achieved by <40% of patients across all treatment arms in advanced therapy RCTs



- CRR not achieved
- CRR achieved

Response graphs are not representative of head-to-head trials and are provided for illustrative purposes only

CNI, calcineurin inhibitor; CRR, complete renal response; DORIS, definition of remission in SLE; RCT, randomized controlled trial; SLE, systemic lupus erythematosus; SRI, SLE Responder Index.

1. Nikfar M, et al. *Int J Clin Pract*. 2021;75(4):e13909. 2. Murimi-Worstell IB, et al. *BMJ Open*. 2020;10(5):e031850. 3. Gomez A, et al. *Front Med*. 2021;8:651249. 4. Refai RH, et al. *Sci Rep*. 2024;14(1):5234. 5. Spies E, et al. *JMIR Form Res*. 2024;8:e52768. 6. Olesińska M, Saletra A. *Reumatologia*. 2018;56(1):45–54. 7. Petri MA, et al. *Ann Rheum Dis*. 2022;81:323. Abstr No. POS0183. 8. Morand EF, et al. *Ann Rheum Dis*. 2023;82(5):639-645. 9. Parodis I, et al. *Arthritis Rheumatol*. 2023;75 (Suppl 9) Abstr No. 2551. 10. Furie R, et al. *N Engl J Med*. 2020;383(12):1117–1128. 11. Rovin BH, et al. *Lancet*. 2021;397(10289):2070–2080. 12. Furie RA, et al. *N Engl J Med*. 2025;392(15):1471–1483.

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B Cells Play a Central Role in the Pathogenesis of SLE

Current therapeutic options often result in incomplete B cell depletion in tissues and lymphoid organs[1]

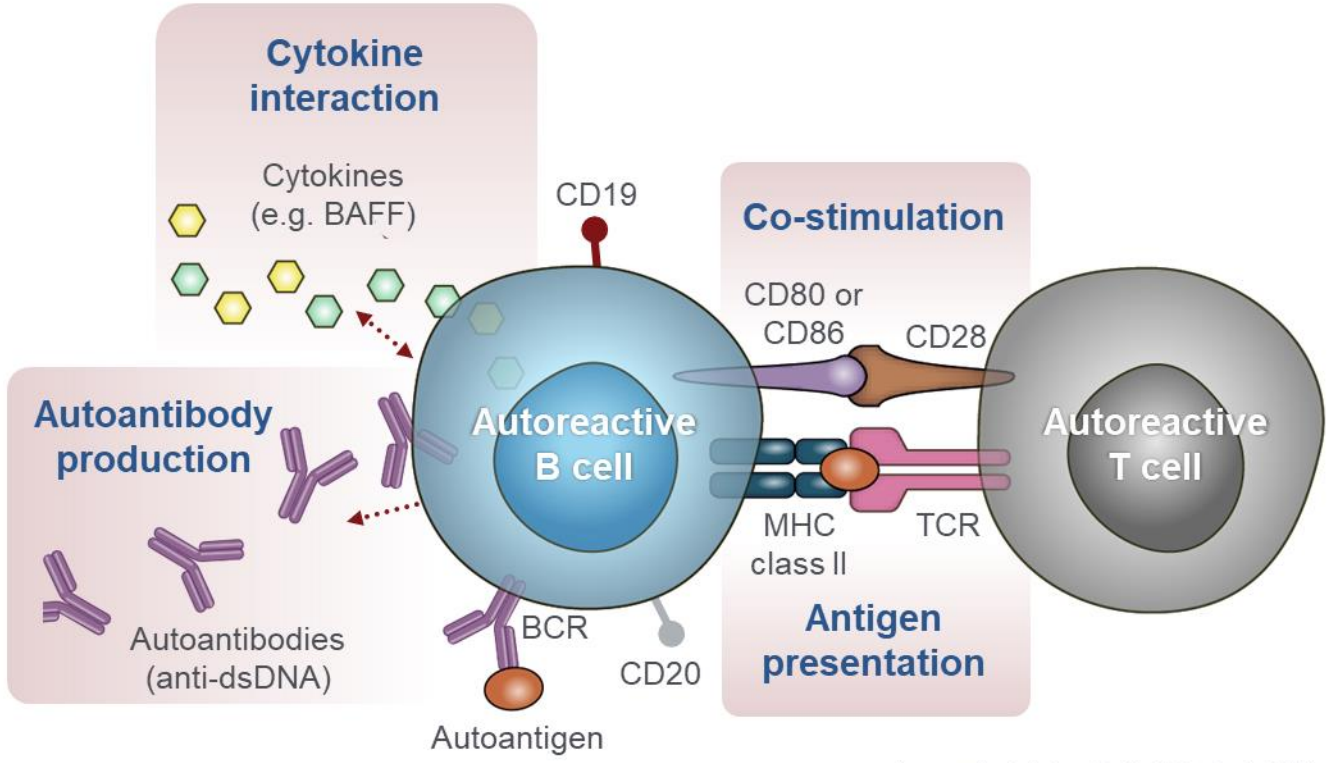

Image adapted from Rubin SJS, et al. 2019[2]

In SLE:

- B cells contribute to autoimmunity through a variety of mechanisms[2,3]
- B cells display profound and multifaceted autoreactivity that extends beyond the bloodstream into inflamed tissues[4]
- B cell-directed therapies are important tools in the treatment of SLE[3]
- Failure to achieve long-standing remission with mAb-based therapy may be due to incomplete B cell depletion[1,5–7]

BAFF, B cell activating factor; BCR, B cell receptor; CD, cluster of differentiation; dsDNA, double-stranded DNA; mAb, monoclonal antibody; MHC, major histocompatibility complex; SLE, systemic lupus erythematosus; TCR, T cell receptor.

1. Schett G, et al. *Ann Rheum Dis*. 2024;83(11):1409–1420. 2. Rubin SJS, et al. *Nat Rev Rheumatol.* 2019;15(5):303–315. 3. Barnas JL, et al. *Curr Opin Immunol.* 2019;61:92–99. 4. Fu Q, Zhang X. *Cell Mol Immunol*. 2021;18(8):2073–2074. 5. Tur C, et al. *Ann Rheum Dis.* 2025;84(1):106–114. 6. Bucci L, et al. *Nat Med*. 2024;30(6):1593–1601. 7. Gonzalez T, et al. *Ann Rheum Dis.* 2024;83(1): 945. Abstr. No. POS1062.

Rese-cel (CABA-201): CD19-CAR T Designed For Autoimmunity

CD19 binder with similar in vitro & in vivo activity to binder used in academic study[1–3]

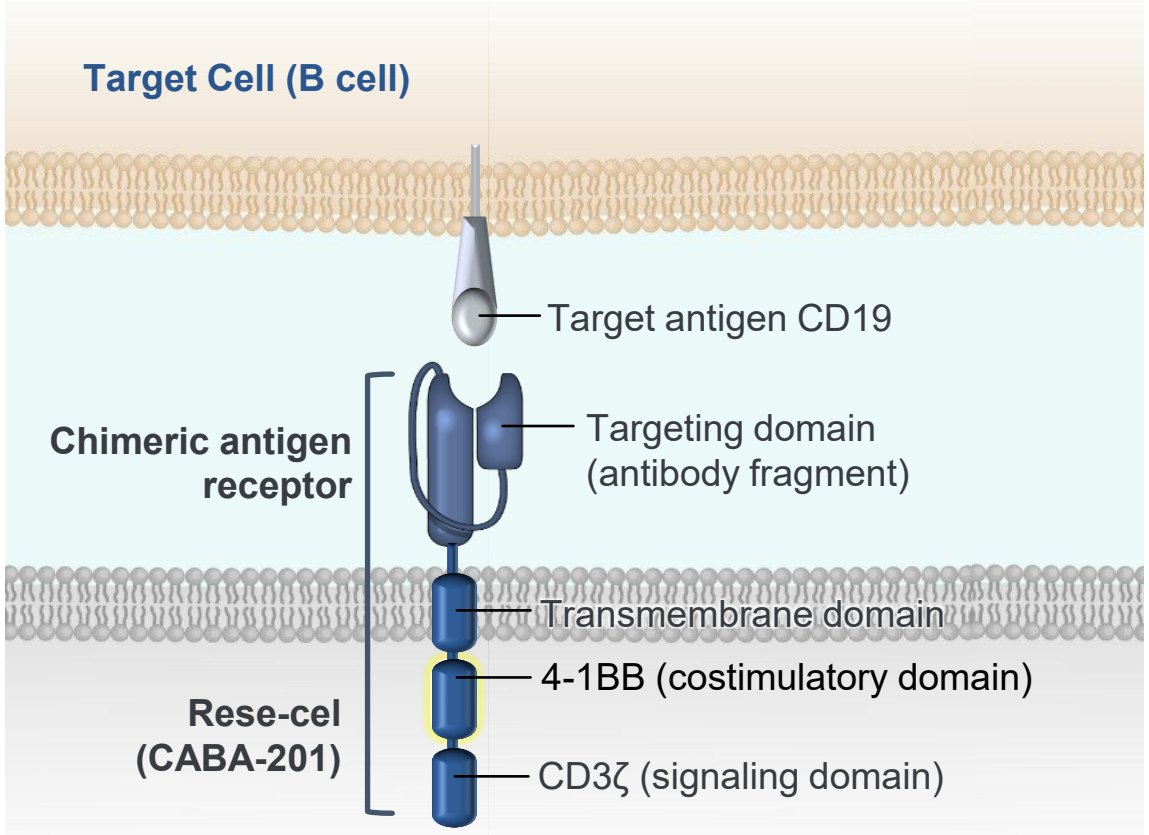

Image adapted from June CH and Sadelain M. 2018.[4]

Rese-cel product design and clinical/translational data

4-1BB costimulatory domain with fully human binder[1]

- Binder with similar affinity and biologic activity to academic FMC63 binder while binding to the same epitopes[1,2]

Same weight-based dose as in academic studies[3,5]

- Potential to provide immune reset based on initial clinical and translational data[5]

Initial patients treated with rese-cel have shown clinical responses with safety data that supports development in autoimmune diseases[6]

CAR, chimeric antigen receptor; rese-cel, resecabtagene autoleucel.

1. Peng BJ, et al. *Mol Ther Methods Clin Dev*. 2024;32(2):101267. 2. Dai Z, et al. *J Cell Physiol*. 2021;236(8):5832–5847. 3. Müller F, et al. *N Engl J Med*. 2024;390(8):687–700. 4. June CH, Sadelain M. *N Engl J Med*. 2018;379(1):64–73. 5. Volkov J, et al. *Mol Ther*. 2024;32(11):3821–3828. 6. Sheikh S, et al. *Arthritis Rheumatol*. 2024;76 (Suppl 9). Abstr. No. 1733.

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Autologous CAR T Therapy: How Rese-cel is Manufactured

Designed to combine antibodies' targeting ability with the cell-killing machinery of a patient's own T cells[1,2]

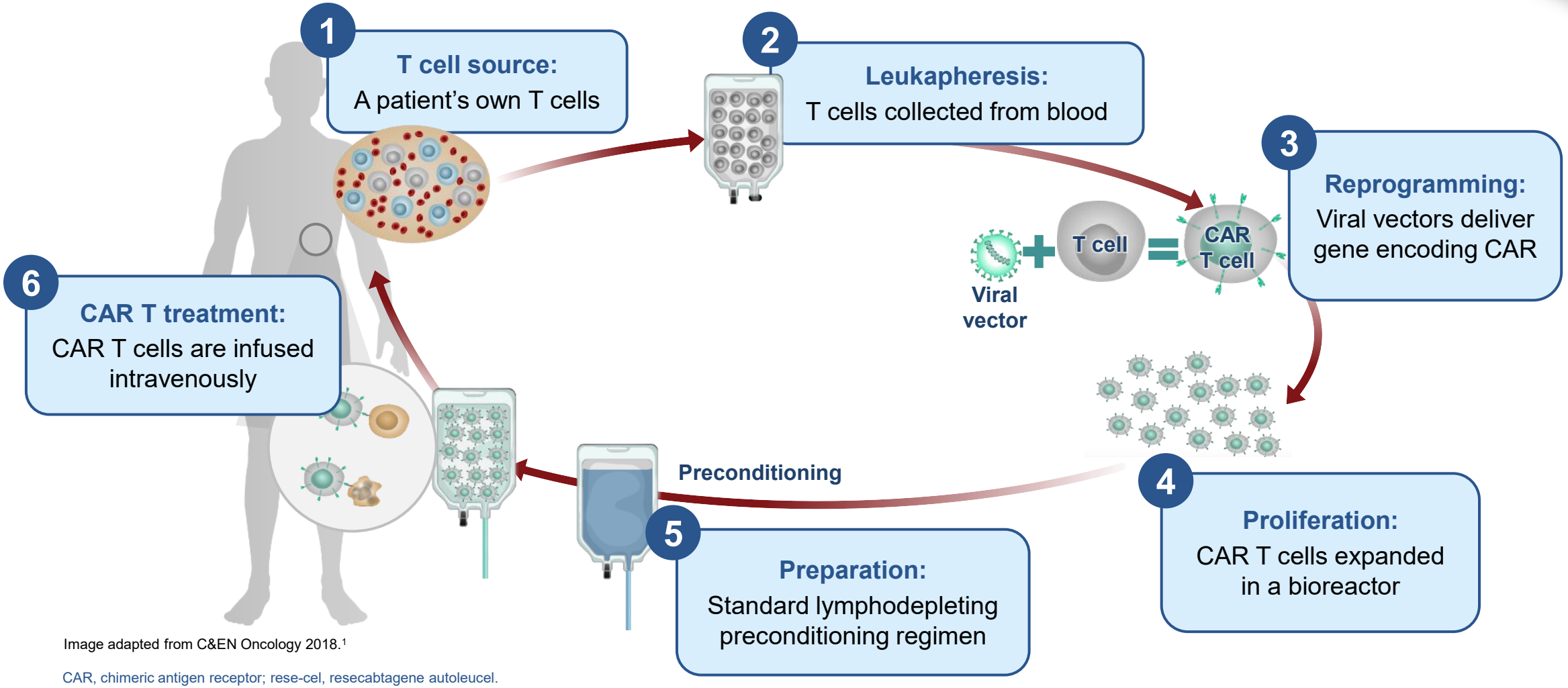


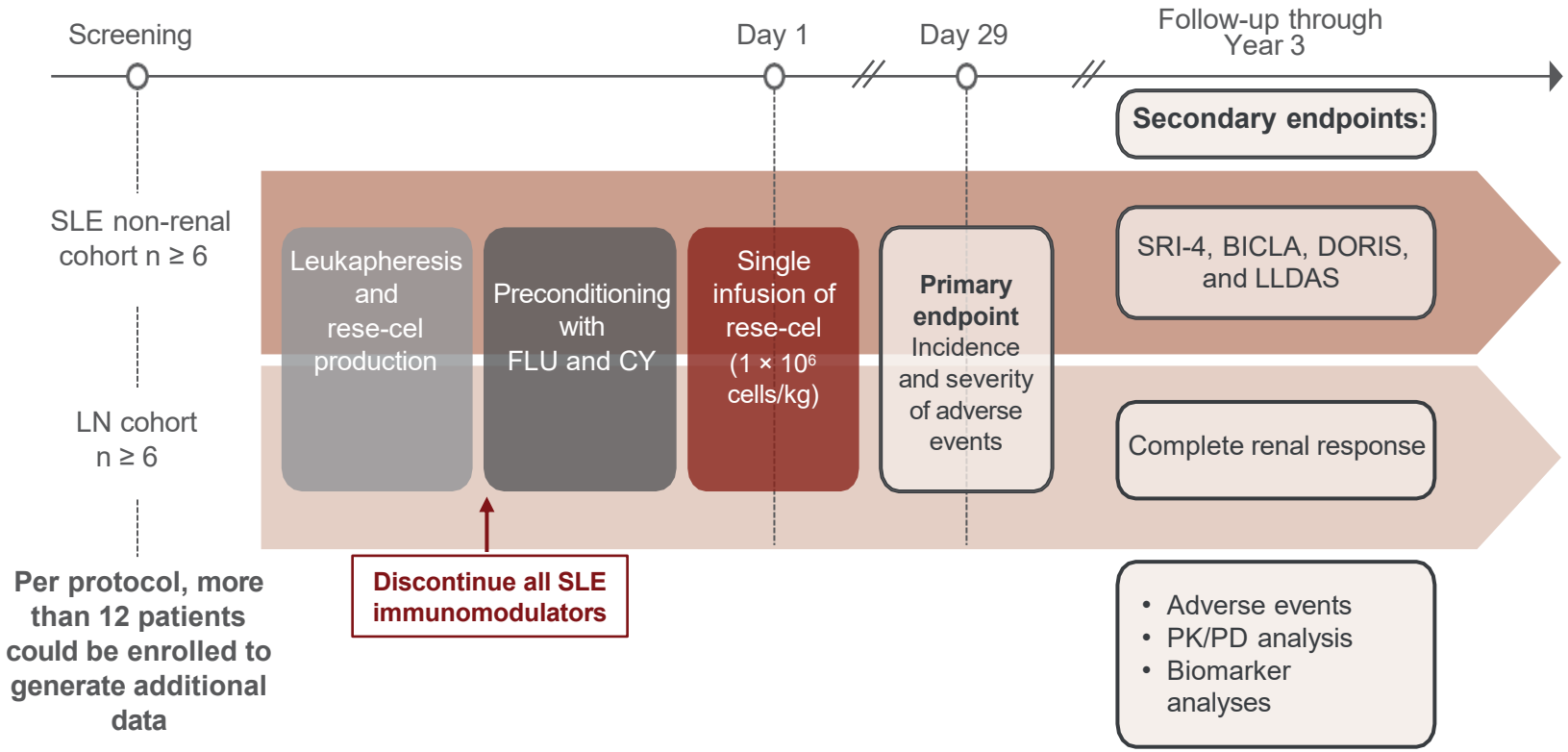
Image adapted from C&EN Oncology 2018.[1]

CAR, chimeric antigen receptor; rese-cel, resecabtagene autoleucel.

1. C&EN Oncology. 2024. Available at: https://cen.acs.org/pharmaceuticals/oncology/Controlling-CAR-T-scientists-plan/96/i19 (accessed May 2025). 2. Peng BJ, et al. *Mol Ther Methods Clin Dev*. 2024;32(2):101267.

RESET-SLE™ Study Design [1,2]

Enrolling patients with active, moderate to severe disease that is refractory to standard of care



Key Inclusion Criteria[1,2]

- Age ≥18 and ≤65 with an SLE diagnosis
- Positive ANA or anti-dsDNA at screening
- Evidence of active disease despite prior or current treatment with standard of care
- **For SLE (non-renal) cohort**: SLEDAI-2K ≥8; pure class V LN patients eligible for this cohort
- **LN cohort**: biopsy-proven LN class III or IV (± class V)

Key Exclusion Criteria[1,2]

- Presence of kidney disease other than LN
- Previous CAR T cell therapy and/or HSCT
- Treatment with B cell-depleting agent within prior ~6 months

ANA, antinuclear antibody; anti-dsDNA, anti-double-stranded DNA antibodies; BICLA, British Isles Lupus Assessment Group-based Composite Lupus Assessment; CAR T, Chimeric Antigen Receptor T cells; CY, cyclophosphamide; DORIS, definitions of Remission In SLE; FLU, fludarabine; HSCT, hematopoietic stem cell transplant; LLDAS, lupus low disease activity state; LN, lupus nephritis; PD, pharmacodynamic; PK, pharmacokinetic; rese-cel, resecabtagene autoleucel; SLEDAI-2K, SLE Disease Activity Index 2000; SLE, systemic lupus erythematosus; SRI, SLE Responder Index.

1. Cabaletta Bio – Data on File. 2. NCT06121297. Available online at: www.clinicaltrials.gov/study/NCT06121297 (accessed May 2025).

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Baseline Characteristics of First 8 Patients in RESET-SLE*

All patients in had active, refractory disease and had failed B cell-targeting therapies

Cohort	Non-renal SLE				LN			
Patient / Cohort	**SLE-1‡**	**SLE-2**	**SLE-3**	**SLE-4**	**LN-1**	**LN-2**	**LN-3**	**LN-4**
Age, sex	26 M	36 F	44 F	37 F	24 F	35 F	26 F	18 M
Disease duration (y)	~6	~17	~9	~10	~2	~8	~16	~4
Autoantibodies§	dsDNA, Sm	dsDNA	dsDNA	dsDNA, Sm	dsDNA, Sm	dsDNA, Sm	Sm	dsDNA
LN class	V	N/A	N/A	N/A	III	IV + V	III + V	IV
SLEDAI-2K†	26	10	8	8	22	14	16	16
UPCR (mg/mg)†	1.08†	N/A	N/A	N/A	7.22	4.85	2.04	1.69
Therapies at screening	HCQ, GC, MMF	GC, AZA	HCQ, MMF, BEL	HCQ**	HCQ, GC, MMF, ANI, VOC	MMF	HCQ, MMF	HCQ, GC, MMF,TAC, BEL
Other prior therapies	CYC, BEL, VOC, TAC	HCQ, MTX, ANI, BEL, MSC, RTX, ADA	GC, MTX	GC, MTX, BEL	BEL, LEF	HCQ, GC, AZA, RTX	GC, MTX, AZA, TOC, UST, RTX, OBI	CYC, RTX, OBI
GC dose at screening (mg/day)	10	7	N/A	N/A**	20	N/A	N/A	5

*As of June 2, 2025.
†Baseline disease activity = activity before preconditioning.
‡Inclusion criteria for LN cohort requires class III/IV LN (± class V).
§All patients were antinuclear antibody positive at screening.
**SLE-4 initiated 20 mg/day of prednisone after screening and before leukapheresis, tapering off by Week 12.
ADA, adalimumab; ANI, anifrolumab; AZA, azathioprine; BEL, belimumab; CYC, cyclophosphamide; dsDNA, double-stranded DNA; GC, glucocorticoid; HCQ, hydroxychloroquine; LEF, leflunomide; LN, lupus nephritis; MMF, mycophenolate mofetil; MSC, mesenchymal stem cell; MTX, methotrexate; N/A, not applicable; OBI, obinutuzumab; RESET, REstoring SElf-Tolerance; RTX, rituximab; SLE, systemic lupus erythematosus; SLEDAI-2K, SLE Disease Activity Index 2000; Sm, Smith; TAC, tacrolimus; TOC, tocilizumab; UPCR, urine protein-to-creatinine ratio; UST, ustekinumab; VOC, voclosporin, y, years.
Cabaletta Bio: Data on File.

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Incidence of Relevant and Related Serious Adverse Events*

Low-grade CRS reported in 2 of 8 patients & ICANS reported in 1 of 8 patients

Cohort	Non-renal SLE				LN			
Patient / Cohort	**SLE-1†**	**SLE-2**	**SLE-3**	**SLE-4**	**LN-1**	**LN-2**	**LN-3**	**LN-4****
CRS†	None	Grade 1	None	None	Grade 1	None	None	None
ICANS†	None	None	None	None	Grade 4	None	None	None
Serious infections‡	None	None	None	None	None	None	None	None
Related SAEs (Grade)§ (Excluding CRS/ICANS)	None	None	None	None	Fever (1) Neutropenic fever (1) Pancytopenia¶ (4)	None	None	None

*As of June 2, 2025; Primary endpoint is incidence and severity of adverse events through Day 29.
†Graded per ASTCT Consensus Grading Criteria. All patients **except** SLE-1 and LN-1 **received** medication for seizure prophylaxis. Tocilizumab was administered for CRS in SLE-2.
‡Coded in System Organ Class of Infections and Infestations and meets seriousness criteria.
§As assessed per US Food and Drug Administration guidelines.
¶Consistent with "Prolonged Cytopenias," which is a labeled warning and precaution for approved oncology CAR T products.
**LN-4: Week 4 safety data presented; efficacy follow up ongoing.
ASTCT, American Society for Transplantation and Cellular Therapy; CAR, chimeric antigen receptor; CRS, cytokine release syndrome; ICANS, immune effector cell-associated neurotoxicity syndrome; LN, lupus nephritis; SAE, serious adverse event; SLE, systemic lupus erythematosus.
Cabaletta Bio: Data on File.

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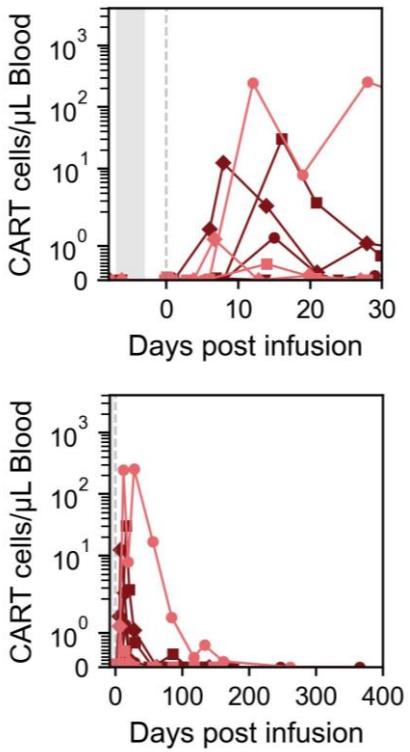
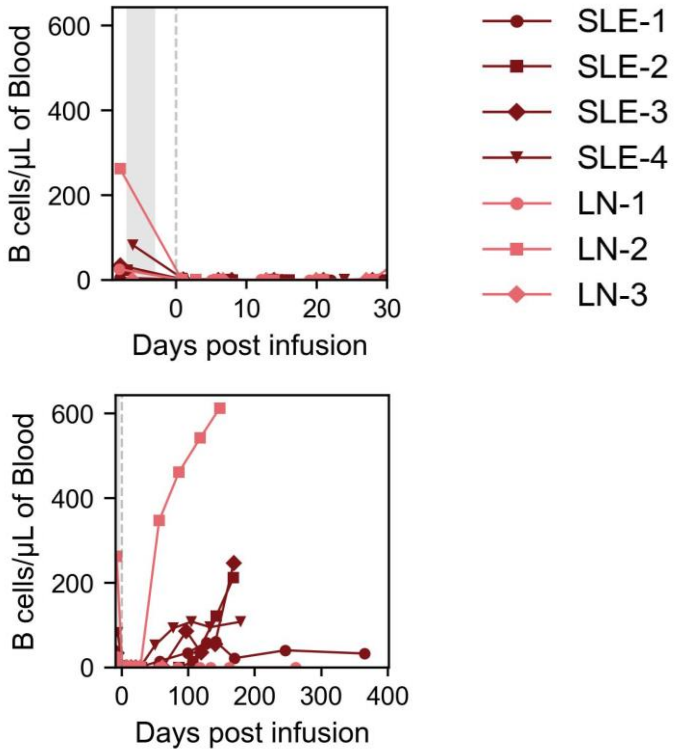
Rese-cel Expansion & B Cell Kinetics

Peak rese-cel expansion and transient peripheral B cell depletion occurred within the first few weeks post infusion

Rese-cel Pharmacokinetics*



B Cell Kinetics



Peripheral B cell repopulation began by 2 to 3 months after rese-cel infusion in SLE and LN* patients

*LN-1 had prolonged rese-cel detection due to TCR activation that corresponded to longer time to B cell repopulation. LN-4: follow up ongoing

CAR, chimeric antigen receptor; LN, lupus nephritis; rese-cel, resecabtagene autoleucel; SLE, systemic lupus erythematosus, TCR, T cell receptor.

Cabaletta Bio: Data on file.

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Efficacy Data Following Rese-cel Infusion*

As of the latest follow up 3 of 4 SLE patients§ achieved DORIS

	Non-renal SLE			
Patient	**SLE-1†**	**SLE-2**	**SLE-3**	**SLE-4**
Latest follow-up	Week 52	Week 32	Week 28	Week 24
DORIS (at latest follow-up)	–§	✓	✓	✓
SLEDAI-2K score‡ (baseline to latest follow-up)	26→12	10→2	8→2	8→2
UPCR (mg/mg) (baseline to latest follow-up)	1.08→1.71	N/A	N/A	N/A
eGFR (mL/min/1.73m^2) (baseline to latest follow-up)	132.7→118.5	N/A	N/A	N/A
CRR (at latest follow-up)	–§	N/A	N/A	N/A
GC-free	✓	✓	✓	✓
IM-free	✓§	✓	✓	✓

*As of June 2, 2025.
†Enrollment in the LN cohort requires class III/IV +/- V LN. SLE-1 had pure class V LN and extra-renal SLE disease activity that met inclusion criteria for the non-renal cohort.
‡SLEDAI-2K components present at latest follow up (SLEDAI-2K contribution score in parenthesis): SLE-1: hematuria (4), proteinuria (4), complement (2), increased DNA binding (2); SLE-2: increased DNA binding (2); SLE-3: increased DNA binding (2); SLE-4: increased DNA binding (2).
§SLE-1 achieved DORIS at Week 48 and CRR at Week 44 and Week 48; on cyclosporine therapy since Week 41 for a non-SLE-related, non-rese-cel-related safety event (macrophage activation syndrome with onset at Week 40).
CRR, complete renal response; DORIS, definition of remission in SLE; eGFR, estimated glomerular filtration rate; GC, glucocorticoid; IM, immunomodulatory; LN, lupus nephritis; N/A, not applicable; rese-cel, resecabtagene autoleucel; SLE, systemic lupus erythematosus; SLEDAI-2K, Systemic Lupus Erythematosus Disease Activity Index 2000; UPCR, urine protein-creatinine ratio.
DORIS = Clinical SLEDAI-2K=0 (irrespective of serology); Physician Global Assessment <0.5; antimalarials; low-dose glucocorticoids (prednisolone ≤5 mg/day); stable immunosuppressives including biologics. **CRR** = UPCR ≤0.5 mg/mg; ≥60 mL/min or no confirmed eGFR decrease of >20% from baseline; no receipt of rescue therapy.
Cabaletta Bio: Data on File.

Efficacy Data Following Rese-cel Infusion*

As of the latest follow up 3 of 4 SLE patients§ achieved DORIS and 1st LN patient achieved CRR

	Non-renal SLE				LN††		
Patient	**SLE-1†**	**SLE-2**	**SLE-3**	**SLE-4**	**LN-1**	**LN-2**	**LN-3**
Latest follow-up	Week 52	Week 32	Week 28	Week 24	Week 44	Week 24	Week 12
DORIS (at latest follow-up)	–§	✓	✓	✓	✓	N/A	N/A
SLEDAI-2K score‡ (baseline to latest follow-up)	26→12	10→2	8→2	8→2	22→0	14→6	16→12
UPCR (mg/mg) (baseline to latest follow-up)	1.08→1.71	N/A	N/A	N/A	7.22→0.24	4.85→2.72**	2.04→2.02
eGFR (mL/min/1.73m^2) (baseline to latest follow-up)	132.7→118.5	N/A	N/A	N/A	72.3→130.9	127.2→125.8	133.2→128.8
CRR (at latest follow-up)	–§	N/A	N/A	N/A	✓	–	–
GC-free	✓	✓	✓	✓	✓	✓	✓
IM-free	✓§	✓	✓	✓	✓	✓	✓

*As of June 2, 2025.
†Enrollment in the LN cohort requires class III/IV +/- V LN. SLE-1 had pure class V LN and extra-renal SLE disease activity that met inclusion criteria for the non-renal cohort.
‡SLEDAI-2K components present at latest follow up (SLEDAI-2K contribution score in parenthesis): SLE-1: hematuria (4), proteinuria (4), complement (2), increased DNA binding (2); SLE-2: increased DNA binding (2); SLE-3: increased DNA binding (2); SLE-4: increased DNA binding (2); LN-2: proteinuria (4), increased DNA binding (2); LN-3: hematuria (4), proteinuria (4), pyuria (4).
§SLE-1 achieved DORIS at Week 48 and CRR at Week 44 and Week 48; on cyclosporine therapy since Week 41 for a non-SLE-related, non-rese-cel-related safety event (macrophage activation syndrome with onset at Week 40).
**LN-2 Week 24 UPCR = 24hr UPCR.
††LN-4: efficacy follow up ongoing.
CRR, complete renal response; DORIS, definition of remission in SLE; eGFR, estimated glomerular filtration rate; GC, glucocorticoid; IM, immunomodulatory; LN, lupus nephritis; N/A, not applicable; rese-cel, resecabtagene autoleucel; SLE, systemic lupus erythematosus; SLEDAI-2K, Systemic Lupus Erythematosus Disease Activity Index 2000; UPCR, urine protein-creatinine ratio.
DORIS = Clinical SLEDAI-2K=0 (irrespective of serology); Physician Global Assessment <0.5; antimalarials; low-dose glucocorticoids (prednisolone ≤5 mg/day); stable immunosuppressives including biologics. **CRR** = UPCR ≤0.5 mg/mg; ≥60 mL/min or no confirmed eGFR decrease of >20% from baseline; no receipt of rescue therapy.
Cabaletta Bio: Data on File.

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Summary from Clinical and Translational Data: RESET SLE™

- Rese-cel was generally well tolerated across 8 SLE & LN subjects treated to date*
  - No CRS in 6 of 8 subjects (Grade 1 in 2 subjects)
  - No ICANS in 7 of 8 subjects (Grade 4 in 1 subject, previously presented)
- Rese-cel provided evidence of efficacy in active and refractory SLE and LN patients†
  - 3 of 4 SLE patients achieved DORIS
    - 4th patient (SLE-1) with pure class V LN
  - LN-1 patient achieved CRR

  } Immunomodulatory and glucocorticoid-free‡
- Rese-cel peak expansion was observed at approximately 2 weeks after infusion
- B cells rapidly and transiently reduced in peripheral blood following rese-cel infusion
  - B cells began to repopulate 2 to 3 months post-infusion
- SLE and LN registrational discussions with FDA scheduled in the third quarter of 2025[1]

*As of June 2, 2025.
† LN-4: efficacy follow up ongoing
‡ SLE-1 achieved DORIS at Week 48 and CRR at Week 44 and Week 48; on cyclosporine therapy since Week 41 for a non-SLE-related, non-rese-cel-related safety event (macrophage activation syndrome with onset at Week 40).
CRR, complete renal response; CRS, cytokine release syndrome; DORIS, definition of remission in SLE; FDA, Food and Drug Administration; ICANS, immune effector cell-associated neurotoxicity syndrome; LN, lupus nephritis; rese-cel, resecabtagene autoleucel; RESET, REstoring SElf-Tolerance; SLE, systemic lupus erythematosus.
1. Cabaletta Bio. (15 May 2025), [Press Release], https://www.cabalettabio.com/news-media/press-releases/detail/128/cabaletta-bio-announces-2027-rese-cel-bla-submission

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- MGH, Massachusetts

Cabaletta Bio team

- Biostatistics
- Clinical Development
- Clinical Operations
- Computational Biology
- Manufacturing
- Medical Affairs
- Quality and Compliance
- Translational Medicine
- Regulatory Affairs